Influence of Acute Physical Activity on Stress Reactivity in Obese and Normal Weight Children: A Randomized Controlled Trial

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Research Article

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Keywords
Childhood obesity · Physical activity · Stress · Cortisol · Blood pressure

Abstract
Objective: Physical activity (PA) may influence acute stress reactivity in children differently depending on their weight. This randomized controlled trial investigated the impact of acute PA and of BMI status (overweight/obese (OB/OW) and normal weight (NW) on stress reactivity. ***Method:*** 50 prepubertal children (24 OW/OB and 26 NW) were randomly assigned to the PA or sedentary arm (SED) for 30 min followed by a stress task. Salivary cortisol, blood pressure (BP), and heart rate (HR) were measured. ***Results:*** An interaction effect between the randomization arms and weight status on salivary cortisol was found after the stress task (p = 0.04). Cortisol increased in the SED, but not in the PA arm (p = 0.004 for differences in time course) of NW children. Time course did not differ between both arms in OW/OB children (p = 0.7). OW/OB SED children had a flat cortisol course, and levels were reduced compared to the NW SED or the OW/OB PA children ($p \leq 0.03$). Systolic BP increased only in the SED arm (p = 0.01).

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HR was higher in the PA than in the SED arm during stress ($p < 0.001$) and showed different time courses ($p = 0.006$). ***Conclusion:*** PA impacted on acute stress reactivity and influenced stress reactivity differently in NW and OW/OB children.

Introduction

Approximately 20% of children in Europe and one-third of children in the US aged 2–19 years are affected by overweight or obesity [1, 2] which carries substantial morbidity [3]. Biological, behavioral, social, and psychological factors contribute to the development of childhood overweight and obesity. Exposure to stress is an important risk factor [4, 5]. Stress, "a state of threatened or perceived as threatened homeostasis", is known to elicit physiological stress responses in the hypothalamic-pituitary-adrenal axis (HPA) and the autonomic nervous system (ANS) [6]. Increased cortisol levels (HPA) in response to a stressor initiate the adaptive function of cognitive processing and mobilizing resources toward tackling the stressor. The ANS triggers the fight-or-flight response by increasing heart rate (HR), constricting blood vessels and increasing blood pressure (BP) [7]. These short-term changes of HPA and ANS activation can be expected under acute stress exposure. However, repeated or extended stress exposure provokes a counter-regulatory long-term response [8].

Obesity in children and adolescents has been associated with more frequent stress exposure such as life events [9, 10] and daily hassles [11] and with changes in counter-regulatory stress responses. This relation can be explained by the effects of the HPA activation under stress conditions on brain reward pathways [12]. Cortisol activates these reward pathways that are related to food addiction [13, 14], resulting in changes of eating behavior including an increase in appetite and a shift to more comfort food preferences; this in turn is related to weight gain [15, 16].

To our knowledge, only two studies have investigated HPA and only few have investigated ANS stress reactivity related to obesity in pre-pubertal children: A blunted cortisol response to stress was associated with increased BMI z-score in preschoolers [17] and in adolescents aged 11–17 years [18]. On the other side, a Chinese study revealed heightened cortisol reactivity in obese children aged 12–13 years [19]. Thus, in children and adolescents, hypo- and hyperreactivity to stress relate to greater BMI cross-sectionally and longitudinally [17–20]. Regarding the ANS system, higher BP reactivity to laboratory stress was associated with a greater abdominal waist and waist-to-hip ratio in adolescents [21, 22] and a greater HR reactivity with percentage body fat following an interpersonal stressor in pre-pubertal children [23]. However, recent studies did not reveal any ANS dysfunction in obese children and adolescents in response to acute stressors [24, 25]. Both heightened and blunted cortisol and cardiovascular stress reactivity can be considered maladaptive, depending on the health outcome [26, 27].

It is known that regular, chronic physical activity (PA) has beneficial effects on cardiovascular disease [28]. It reduces the HPA reactivity to acute stress [29, 30] and reduces BP [31–33], and HR reactivity in adults [32]. This is important, as heightened stress reactivity predicts hypertension and future cardiovascular disease [26, 27]. Regular PA is also related to improved mood/reduced anxiety in children and young adults [34–36]. A reduction in stress reactivity might contribute to the role of PA as a protective factor in stressful experiences. So far, only two studies investigated the effects of PA on stress reactivity in normal-weight (NW) children. Chronic PA was related to lower diurnal HPA activity and lower HPA reactivity to acute stress in pre-pubertal children [37]. Further, acute PA decreased BP but not HR reactivity to interpersonal stress in pre-pubertal schoolchildren with healthy body weight [38].

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Whether acute PA has a similar beneficial effect on the HPA axis is unknown. It is also unclear whether acute PA modulates the HPA and ANS stress reactivity in obese children, whose baseline values might differ and who are likely to be frequently exposed to stressful social situations [39].

Therefore, the main aims of the current study were to investigate i) the role of PA in modulating stress reactivity of the HPA axis (salivary cortisol) and the ANS (BP and HR) after acute social stress exposure in overweight/obese (OW/OB) and NW pre-pubertal children and ii) whether stress reactivity differed between OW/OB and NW children. In a second step, we also evaluated if OW/OB children had more frequent exposure to negative life events and early parental separation compared to NW children, as these might contribute to differences in acute stress reactivity.

Material and Methods

Participant Consent and Recruitment

Flyer advertisements and advertisements on the University Hospital Lausanne website were employed to recruit NW and OW/OB children from the general population between September 2012 and March 2013. In parallel, 7- to 11-year-old OW/OB children were consecutively identified by clinicians working at the Children's Hospital Lausanne and in the pediatric and emergency departments at the University Hospital Lausanne. Their parents who had agreed to be contacted subsequently received a phone call by the research team to verify their interest and to check that their children fulfilled the inclusion criteria: i) BMI ≥ 90th percentile for the OW/OB or BMI < 90th for the NW children according to WHO criteria [40]; ii) aged between 7 and 11 years; iii) reported absence of puberty and of chronic medical problems, such as epilepsy and asthma; iv) basic knowledge of French; and v) the child's physical ability to perform a running exercise. An individual appointment was arranged after written parental consent was obtained. Each family received a gift voucher of CHF 50.00 for their participation.

A total of 30 NW children opted in to participate but 4 did not attend the appointment (due to illness or having forgotten the appointment). The parents of 52 OW/OB children were contacted, 38 agreed to participate but 14 subsequently did not attend the appointment. The final sample consisted of 24 OW/OB (5 OW and 19 OB) and 26 NW children.

This study was part of a clinical trial (clinicaltrials.gov NCT01693926) investigating the impact of PA on stress reactivity and food intake/choices in children [41]. Ethical approval was provided by the ethics committee of the canton Vaud (Switzerland, protocol 286/2012).

Study Design and Measures

Study Design

Figure 1 provides an overview of the study. Individual appointments took place between 16.00 h and 19.00 h at the University Hospital Lausanne. Given the length of the experiment, parents were advised that their children should have a fruit snack at 15.00 h. Eating during the experiment was not allowed. At the beginning of the assessment, a physical exam was performed, and parents then completed a demographic questionnaire and an interview with a psychologist focusing on major life events in a separate room. Afterwards, the children were separated from their parents and stayed with the research team for the rest of the study. After stratification for weight status, all children were randomly allocated (1:1, using a computer-generated numbers by a staff member not involved in the activities or testing) to a moderate PA (n = 13 NW children and n = 12 OW/OB children) or a sedentary activity arm (n = 13 NW children and n = 12 OW/OB children) for 30 min. Group allocation was concealed until the start of the activity.

In the moderate PA arm, children engaged in playful exercises with a basketball that included coordination, balance and speed with a physical education (PE) specialist during 30 min. This was followed by a running exercise, involving a slalom using the ball and a basketball net, a short running competition against the PE specialist, and going up and down the stairs. The PE specialist ensured that the children's perceived exertion was rated as "somewhat hard to hard" [42]. In addition, HR was monitored throughout the intervention by a polar watch, aiming for a HR of 140 beats/min for the NW and 160 beats/min for the OW/OB children [43]. This was done to correct for the increased oxygen consumption given the higher exercise

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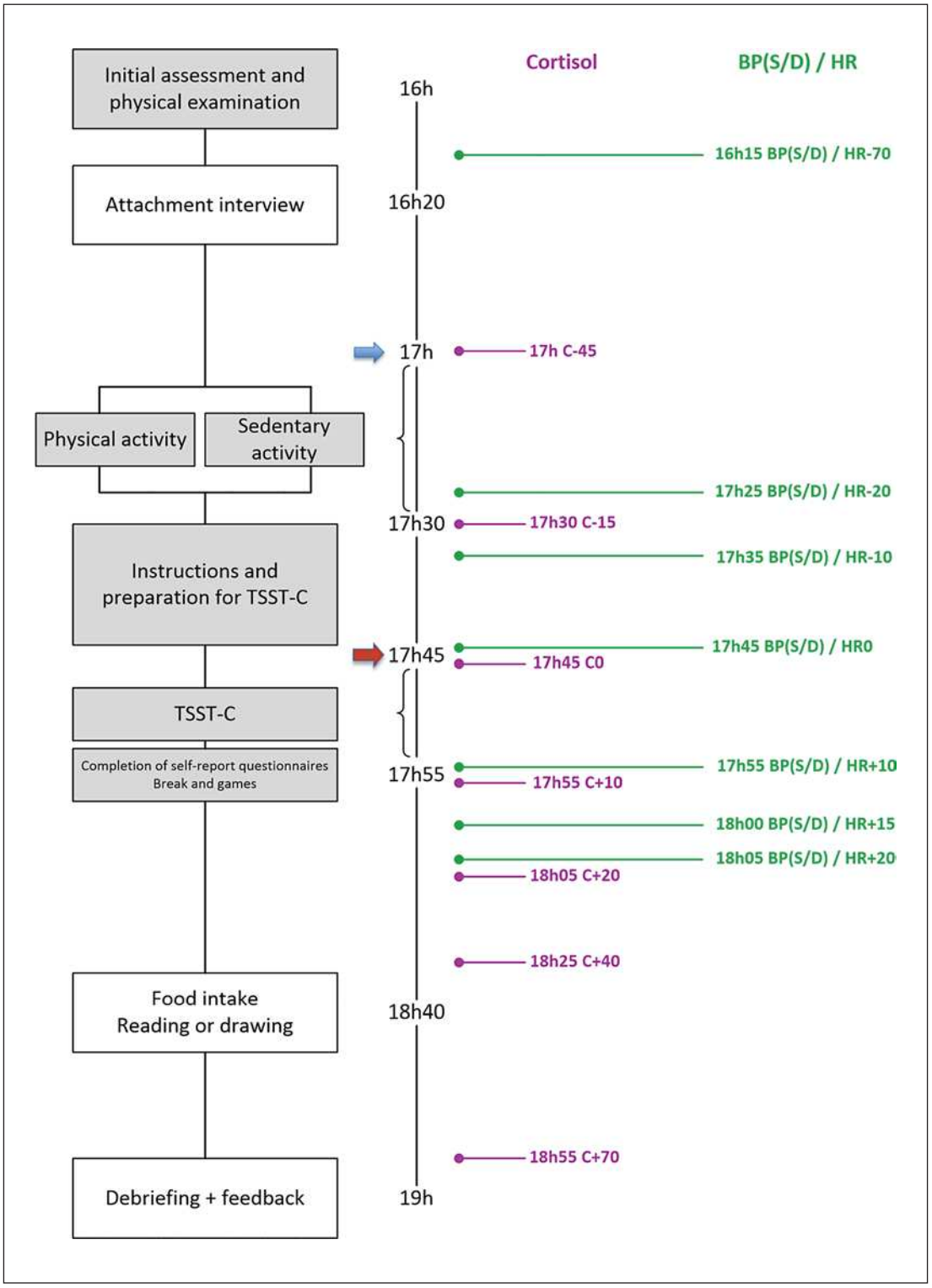

Fig. 1. Study design including repeated measurements of heart rate, blood pressure and salivary cortisol. C, salivary cortisol (timing related to beginning of TSST-C); BP, blood pressure; HR, heart rate; PA, physical activity; SA, sedentary activity; TSST-C, Trier Social Stress Test for Children.

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intensity in the OW/OB group [44]. The PE specialist also ensured that the children's perceived exertion was rated as "somewhat hard to hard" based on a repeated check every 5 min that rating on the categorized Borg scale was between 4 and 6 [45]. Children assigned to the control arm (sedentary activity, SED) chose between playing calm board games, reading books, or drawing in the presence of the PE specialist during 30 min. In the meantime, parents completed self-report demographic questionnaires (see below).

During the 15-min recovery period following this, all children in both arms received instructions for the Trier Social Test for Children (TSST-C) [37, 46]. The test has been developed to induce psychosocial stress and has been shown to elicit a strong and reliable stress response [46]. This standardized procedure consists of a 3-min preparation period, followed by a 5-min speech task and a 5-min mental arithmetic task (both tasks performed in front of an audience of two experts and were video-taped) adapted to the age and performance of the child.

After the stress test, children were brought to the kitchen, where they were left alone for 20 min and were told that they could read, color in pictures and/or eat freely from a buffet. In addition, they had coloring material, comic books, and games at their disposal. Following this, all parents and children were debriefed on the study procedure.

Measures

Children's Measures

Anthropometric Measures. Body weight was measured in light clothes and without shoes to the nearest 0.1 kg with a digital medical scale. Standing height was assessed without shoes to the nearest 0.1 cm with a stadiometer. Children's BMI was calculated and their attribution to the weight category confirmed (BMI ≥90th percentile for the OW/OB or BMI <90th for the NW [40]. Waist circumference was measured by a flexible tape midway between the iliac crest and the lowest border of the rib cage.

Stress Reactivity (Fig. 1). HR, systolic BP (S) and diastolic BP (D) were measured using a sphygmomanometer (Omron 705-IT). The inflatable cuff was placed 2.5 cm away from the elbow whilst the child was sitting down. Two subsequent measurements were taken, and the mean was used to achieve maximum accuracy. Measurements for S, D, and HR were taken at seven different time points (Fig. 1): 70 min (baseline values, S/D/HR-70), 20 min (S/D/HR-20), 10 min (S/D/HR-10) before the start of the TSST-C, at the start of the TSST-C at 17.45 h (S/D/HR0), and 10 min (S/D/HR+10), 15 (S/D/HR+15), and 20 min (S/D/HR+20) after the start of the TSST-C. Salivary cortisol was measured using Salivette (Sarstedt, Nümbrecht, Germany) collection devices using the passive drool method at seven different time points: 45 min (baseline value, C-45) and 15 min (C-15) before the start of the TSST-C, at the start of the TSST-C (C0), and 10 min (C+10), 20 min (C+20), 40 min (C+40), and 70 min (C+70) after the start of the TSST-C. S/D/HR-20 corresponded to the last 5 min of the physical/sedentary activity, and C-15 corresponded to the end of the physical/sedentary activity and before the instructions/preparation for the TSST-C. After collection, cortisol samples were stored at –20 °C until badge analysis. Analyses took place at the Technical University of Dresden (Germany) using a commercial chemiluminescence immunoassay (CLIA; IBL Hamburg, Germany).

Parents' Measures

Demographic Information. Parents completed a brief demographic questionnaire including their place of birth, educational level, and current professional activity. Parental migrant status was assigned if at least one parent was born outside of Switzerland [47], parental socio-economic status was calculated based on maternal/paternal education (1 = primary education, no professional training to 4 = university degree) and current professional activity (1 = unqualified employment to 4 = managing director or independent academic), with a maximum total score of 4 [48].

Major Life Events. Parents participated in a structured interview with a Master level psychologist during which they were asked whether they had been exposed to 13 potential major life events (such as death of someone they were close to, history of abuse, serious accident, divorce, etc.) [9, 49, 50]. For each major life event, participants had to indicate when it had occurred and whether they or anyone else in their family had been involved, and they were asked to rate how much stress this had caused them as well as their child (perceived parental and child stress, Likert scale; 1 = no stress; 3 = high stress) [9]. They then had the opportunity to add other major life events not included in the list.

Furthermore, parents were asked whether their child had experienced periods of early separation from them before the age of 3 years.

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Table 1. Baseline demographic and anthropometric characteristics and stress exposure by randomization arms and weight status

Outcome variables	Normal weight (*n* = 26)		Overweight/obese (*n* = 24)		Randomization arms (*p* value)	Weight status (*p* value)
	sedentary (*n* = 13)	physical activity (*n* = 13)	sedentary (*n* = 12)	physical activity (*n* = 12)		
Age, years	8.5±0.9	8.6±0.6	9.6±1.4	8.9±1.2	0.40	0.04
Girls/Boys, *n*	7/6	6/7	6/6	9/3		
Height, cm	134.4±6.7	131.9±5.6	143±12	137.8±11.4	0.18	0.008
Weight, kg	28.5±3.9	28.4±3.8	50.4±9.2	43.6±10	0.33	<0.0001
BMI, kg/m^2	15.7±1.1	16.2±1.2	24.6±3.3	22.7±2.6	0.62	<0.0001
BMI z-score	–0.15±0.5	0.14±0.7	3.13±1.5	2.56±1.1	0.81	<0.0001
Parental SES	2.84±0.6	3.11±0.6	2.25±0.8	2.5±0.2	0.22	0.003

BMI, body mass index; parental SES, socioeconomic status of the parents. Randomization arm = physical activity versus sedentary activity; Weight status = overweight/obese versus normal weight. Data are shown as mean ± standard deviation. Unadjusted differences between weight status and randomization arms are calculated by unpaired *t* test or chi2 test, as appropriate.

Data Analysis

Power analysis was done for D reactivity as no previous study had assessed the impact of acute PA on cortisol stress reactivity in children. If the effect of PA on the average stress reactivity of D were of the order of one standard deviation (5.6 mm Hg), which would be consistent with previous research [38], then 23 children per group would be sufficient to provide a corresponding result of statistical significance at the 5%-level with a probability of 90%.

Qualitative data were described using absolute and relative frequencies and quantitative data using mean ± standard deviation or median and interquartile range, as appropriate. Differences between randomization arms (physical vs. sedentary activity) or weight status (OW/OB vs. NW children) in baseline demographic and anthropometric characteristics were assessed using an unpaired *t*-test, and differences in exposure to potential stressors and perceived stress using the chi-square test. The stress reactivity measures (cortisol, S, D, and HR) were ln-transformed before analysis to improve the distributional properties of their residuals. Unadjusted differences between randomization arms or weight status in measures of stress reactivity at the fixed time points were assessed using unpaired *t*-test (Table 1).

Adjusted differences between randomization arms or weight status in measures of stress reactivity at baseline or at the start of the TSST-C were assessed using linear regression models additionally including age, sex, and parental SES as well as baseline values of the respective variables. Changes in measures of stress reactivity over time either from study begin or from the start of the TSST-C were analyzed using mixed linear models with time as a factor variable and a random intercept for each child, adjusting for age, gender, and parental SES. Group differences in mean values of stress reactivity measures over time are also reported. The main aims, i.e. differences in the time course of the respective outcome according to weight status or type of intervention, were assessed using interaction terms of the two factors with the factor time and by stratifying analyses according to them. Interactions between all three factors were used to assess differences in the time course of the groups. To simplify comparisons, the time course under the intervention was also summarized by averaging outcome levels across the time points after the intervention phase. All analyses relating to the time course after intervention were done with adjustment for potential differences in the outcome level at the last time point before intervention. As we found significant differences in the time course of cortisol level according to weight status and type of intervention, we also calculated the area under the curve to the ground (AUCg) [51] between the time point at the start of the intervention (at C0) and the last measurement at C+70 for cortisol. This measure was log-transformed for analysis, and adjustment for potential differences before the intervention was achieved using the natural logarithm of the rectangular area defined by the natural

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logarithm of cortisol level at the start of the intervention and the length of the time interval between the start of the TSST-C and the last measurement. Results of the regression analyses are reported as percentage changes in geometric means (with 95% confidence interval (95% *CI*)). In the following, the term mean will be used for geometric mean for the sake of simplicity. Statistical significance was defined at the usual 5% level for main effects and at the 10% level for interactions. All analyses were performed using STATA version 12.0 (Stata Corp, College Station, TX, USA).

Results

Baseline Sample Characteristics

The OW/OB and NW groups differed with regards to age, anthropometric measures, and parental SES (all $p < 0.04$; Table 1). There were no significant differences between the PA and sedentary control arm within NW and OW groups (all p = NS). There were no significant group differences regarding the number of negative life events (OW/OB: 5.2 ± 1.9 vs. NW: 6.1 ± 2.8, $p = 0.2$) or the number of early separation (OW/OB: 13/11 vs. NW: 8/18, $p = 0.2$) and no differences between the randomization arms with regards to the number of negative life events (all p = NS). Further, there was also no difference in the presence of early separation events from parents (all p = NS) but even a tendency for the perceived stress (by parents or children) caused by the life events to be lower in the OW/OB (compared to the NW children ($p = 0.05$ and 0.08, respectively)).

The mean values of cortisol, BP or HR over the entire sample all changed from baseline to the end of the testing period (i.e. +70 min for cortisol; +20 min for the other values, all $p \leq 0.03$).

Salivary Cortisol Reactivity

Impact of Preceding PA on Cortisol Reactivity after the TSST-C

The time course of cortisol showed significant differences between the randomization arms (PA vs. sedentary activity) and the weight status (OW/OB vs. NW children) (Fig. 2a, p for interaction = 0.045). Similar differences between activity arms and weight status were observed when using the AUCg of the cortisol concentrations after the TSST-C and adjusting for pre-existing differences between the two arms or weight status groups in the level of cortisol at the start of the TSST-C (p for interaction = 0.035). Therefore, the impact of PA on cortisol reactivity after the TSST-C was analyzed separately according to the weight status group.

In the NW group, the time course of cortisol was significantly different between the two activity arms ($p = 0.004$): In the NW sedentary arm, there was a tendency to an increase in average cortisol after the TSST-C ($p = 0.07$) with the mean value over the four measurements 10–70 min after the TSST-C (C+10 to C+70) being 34% higher than at the start (C0; $p = 0.04$, 95% *CI:* 1–79%). In contrast, in the NW physically active arm, there was a decrease in average cortisol ($p = 0.009$) with the mean value over the four measurements 10–70 min after the TSST-C (C+10 to C+70) being 26% lower than at the start (C0; $p = 0.003$, 95% *CI:* 10–40% lower).

Regarding differences in cortisol concentrations, the adjusted mean cortisol levels were 58% higher in the PA compared to the sedentary arm at the start of the TSST-C (C0; $p = 0.04$; 95% *CI:* 4–140%), but there were no differences in the mean levels between both activity arms over the 70 min after the TSST-C ($p = 0.9$).

In the OW/OB group, the time course of cortisol was not different between the two activity arms ($p = 0.7$) and over time cortisol levels did not change ($p = 0.9$ for the sedentary and $p = 0.4$ for the PA arm, respectively). Regarding differences in cortisol concentrations, the

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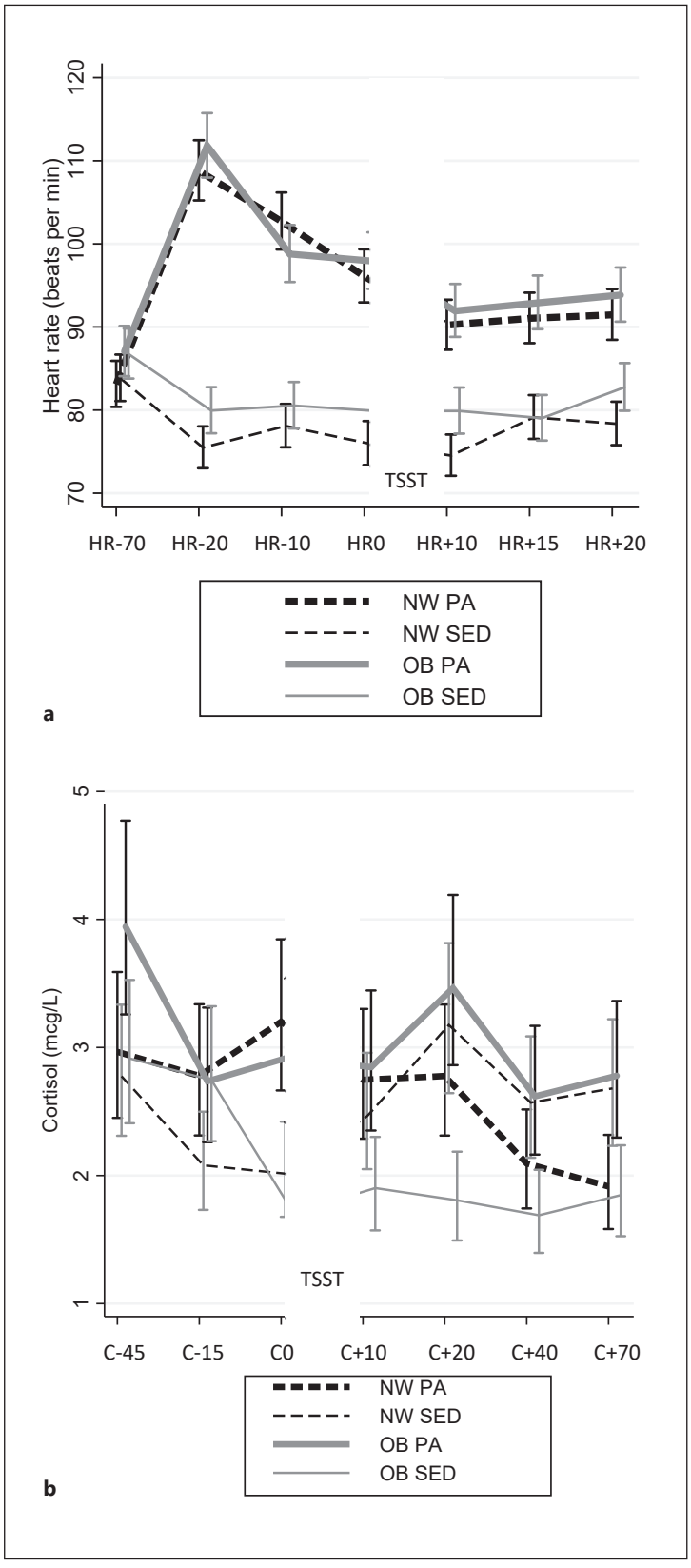

Fig. 2. a–d: Repeated measurements of heart rate for the four subgroups (OW/OB, overweight/obese; NW, normal weight; PA, physical activity; SED, sedentary activity; TSST-C, Trier Social Stress Test for Children. The time points show the beginning of the activities for cortisol (C) at –45 min before the start of the TSST-C (C-45) and repeated assessment at 15 min before TSST-C (C-15), at the start (C-0) and after the TSST-C at different time points including 10 min (C+10), 20 min (C+20), +40 min (C+40) and 70 min (C+70). For systolic (S) and diastolic blood pressure (D) and heart rate (HR) arrows show the beginning of the activities at 70 min before the stress task (S-70, D-70, PHR-70) and repeated measures before the stress task at 20 min (S-20, D-20, HR-20) and at 10 min (S-10, D-10, HR-10), at the start (S0, D0, HR0) and after the TSST-C at different time points including 10 min (S+10, D+10, HR+10), 15 min (S+15,D+15,HR+15) and at 20 min (S+20, D+20,HR+20). Data are shown as geometric means and 68% confidence intervals.

(Figure continued on next page.)

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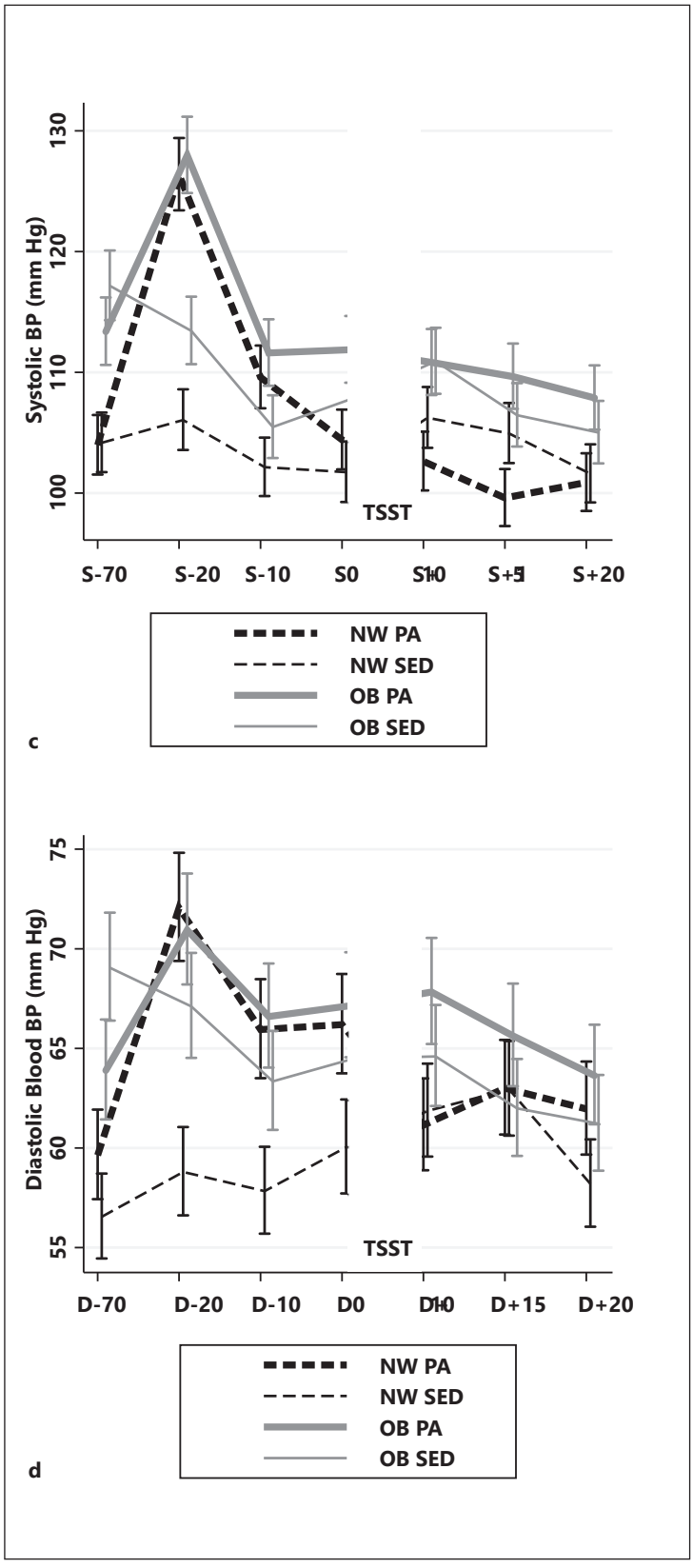

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adjusted mean cortisol levels were 95% higher in the PA compared to the sedentary arm at the start of the TSST-C (C0; $p = 0.04$, 95% *CI:* 2–272%) and the mean levels in the PA arm remained 80% higher over the 70 min after the TSST-C ($p = 0.007$, 95% *CI:* 17–177%).

Impact of Weight Status on Cortisol Reactivity after the TSST-C

The time course after the TSST-C between the two weight status groups did not differ in the sedentary ($p = 0.58$) or the PA arm ($p = 0.1$).

Cortisol concentrations at the start of the TSST (C0) did not differ between NW and OW/OB in either activity arm ($p \geq 0.4$; Table 2 and Fig. 2a show unadjusted levels). In the sedentary arm, the mean cortisol levels over the 70 min after the TSST-C were 49% lower in the OW/OB compared to the NW group ($p = 0.028$; 95% *CI:* 7–72%), but they did not differ in the PA arm ($p = 0.2$).

Cortisol Reactivity before the TSST-C

Adjusted baseline cortisol values before the physical or sedentary activity (17.00 h; C-45) did not differ between weight status groups but were higher in the PA compared to the sedentary arm ($p = 0.04$; Table 2 and Fig. 2a show unadjusted levels). Between the beginning of the activity (C-45) and the beginning of the TSST-C (C0), salivary cortisol decreased in both weight status groups ($p \leq 0.004$), was unchanged in the NW PA arm ($p = 0.5$) and even decreased in the OW/OB PA arm ($p = 0.03$).

BP and HR

Impact of Preceding PA on BP and HR Reactivity after the TSST-C

The time course of the BP or HR values after TSST-C showed no significant differences between the randomization arms (PA vs. sedentary activity) and the weight status (OW/OB vs. NW children) (p for interaction ≥0.3). Thus, the impact of PA was analyzed together for NW and OW/OB children.

The time course of the D did not differ between the two activity arms ($p = 0.4$). There was some indication of a different impact of PA on the time course of the S ($p = 0.09$). S increased in the sedentary arm ($p = 0.01$) with levels at S+10 (end of the TSST-C), being higher compared to baseline ($p = 0.03$). On the other side, in the PA arm S tended to decrease ($p = 0.06$), with the mean value over all 3 measurements 10–20 min after the TSST-C (S+10, S+15, S+20) being 3% lower compared to S0 ($p = 0.03$, 95% CI 0.2–5%). Regarding differences in BP values, there were no differences in the S, while D tended to be higher in the PA arm ($p = 0.1$ and 0.058, respectively; Table 1 and Fig. 2b/c show unadjusted values). There were no differences in the mean BP values between both arms over the 20 min after the TSST-C (both $p > 0.1$).

However, the time course of the HR differed between the two activity arms ($p = 0.006$). In the sedentary arm, there was a tendency for an increase of HR after the TSST-C ($p = 0.06$) with the value 20 min after the TSST-C (HR+20) being higher compared to HR0 ($p = 0.06$) while HR decreased in the PA arm ($p = 0.008$), with the mean value over the 3 measurements 10–20 min after the TSST-C (HR+10 to HR+20) being 5% lower than HR0 ($p < 0.001$, 95% *CI:* 3–8%). Regarding differences in HR values, the adjusted HR was 25% higher in the PA compared to the sedentary arm ($p < 0.001$, 95% *CI:* 17–33%; Table 2 and Fig. 2d show unadjusted values) and the mean HR values over the 20 min after the TSST-C remained 19% higher ($p < 0.001$, 95% *CI:* 12–26%).

Impact of Weight Status on BP and HR Reactivity after the TSST-C

There was no difference in the time course between the two weight status groups ($p = 0.9$). Neither D or HR nor their respective time course differed between the two weight status groups after TSST-C (all $p \geq 0.1$), but S at the start of the TSST-C was 5% higher in the OW/

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Table 2. Stress reactivity of salivary cortisol, systolic and diastolic blood pressure and heart rate for the four subgroups

Stress reactivity	Normal weight (n = 26)		Overweight/obese (n = 24)		Randomization arms (p value)	Weight status (p value)
	sedentary (n = 13)	physical activity (n = 13)	sedentary (n = 12)	physical activity (n = 12)		
Salivary cortisol (C)						
45 min before TSST-C (C-45)	3.8 (1.9–4.1)	3.3 (2.6–3.9)	3.3 (2.6–3.9)	3.8 (3.0–5.0)	0.2	0.25
15 min before TSST-C (C-15)	2.4 (2.0–2.9)	2.8 (2.4–3.2)	3.1 (2.0–3.8)	3.2 (2.3–3.8)	0.23	0.29
Start of TSST-C (C0)	2.1 (1.5–2.8)	3.1 (2.2–4.6)	1.9 (1.2–2.9)	3.1 (2.2–4.8)	0.006	0.5
10 min after start of TSST (C+10)	2.1 (1.6–4.5)	2.9 (1.5–4.7)	2.4 (1.2–3.0)	3.1 (1.9–4.0)	0.18	0.55
20 min after start of the TSST (C+20)	2.6 (2.2–8.7)	2.7 (1.7–5.8)	2.1 (1.4–3.1)	3.4 (2.4–5.9)	0.29	0.45
40 min after start of the TSST (C+40)	2.2 (1.5–7.2)	2.0 (1.4–3.8)	1.8 (1.4–2.8)	2.7 (1.5–4.9)	0.67	0.68
70 min after start of the TSST (C+70)	3.2 (1.9–4.9)	2.6 (1.8–3.0)	1.9 (1.5–2.5)	2.9 (2.1–3.4)	0.89	0.97
Systolic blood pressure (S)						
70 min before TSST-C (S-70)	108.5 (95.0–112.8)	104.0 (97.8–112)	116.3 (108.4–122.8)	112.3 (109.3–121.0)	0.59	<0.0006
20 min before TSST-C (S-20)	104.5 (103–113.3)	131.0 (119.0–134.0)	112.0 (104.1–121.9)	124.5 (121.4–138.9)	<0.0001	0.23
10 min before TSST-C (S-10)	103 (99.0–106.5)	110.0 (105.3–112.8)	106.5 (97.8–110.4)	110.8 (103.0–125.1)	0.005	0.29
Start of TSST-C (S0)	101.5 (95.4–109.2)	105.5 (100.0–108.7)	108.0 (103.8–114.0))	112.8 (104.5–119.0)	0.22	0.007
10 min after start of TSST-C (S+10)	105.0 (102.5–112.8)	102 (98.3–109.3)	111.3 (104.6–113.9)	109.8 (103.4–120.3)	0.46	0.01
15 min after start of TSST-C (S+15)	106.5 (101.8–111.0)	101.0 (95.8–103.8)	104.5 (100.0–114.3)	108.3 (102.0–116.9)	0.63	0.04
20 min after start of TSST-C (S+20)	103.5 (95.0–107.8)	101.5 (95.5–104.3)	104.5 (97.6–111.6)	108.0 (101.1–117.0)	0.68	0.02
Diastolic blood pressure (D)						
70 min before TSST-C (D-70)	56.5 (49.5–62.5)	58.5 (55.3–66.8)	68.8 (54.3–77.6)	57.5 (65.3–70.3)	0.86	0.01
20 min before TSST-C (D-20)	57.0 (52.0–64.3)	71.0 (68.3–76.3)	64.0 (60.1–79.5)	72.3 (66.0–78.1)	0.002	0.2
10 min before TSST-C (D-10)	57.0 (52.5–61.5)	67.0 (62.0–71.3)	62.3 (57.5–66.6)	66.6 (61.6–74.8)	0.009	0.17
Start of TSST-C (D0)	58.3 (52.3–67.9)	66.0 (62.3–72.3)	62.5 (62.0–66.8)	66.8 (62.0–69.9)	0.06	0.27
10 min after start of TSST-C (D+10)	61.0 (51.5–72.8)	62.0 (55.8–67.5)	64.0 (60.8–67.5)	67.0 (63.0–73.0)	0.65	0.05
15 min after start of TSST-C (D+15)	62.5 (56.8–72.0)	63.5 (57.5–66.8)	61.3 (55.6–67.6)	64.3 (61.8–69.0)	0.43	0.72
20 min after start of TSST-C (D+20)	58.0 (51.0– 66.8)	60.5 (57.5–68.3)	61.0 (56.0–67.6)	62.5 (59.1–67.1)	0.12	0.24
Heart rate (HR)						
70 min before TSST-C (HR-70)	81.0 (77.0–87.8)	82.0 (75.5–91.5)	90.8 (76.9–95.6)	86.0 (82.1–93.3)	0.92	0.01
20 min before TSST-C (HR-20)	76.5 (70.5–81.5)	106.5 (95.8–123.8)	83.5 (70.1–89.8)	117.0 (97.9–121.5)	<0.0001	0.51
10 min before TSST-C (HR-10)	76.5 (72.8–88.5)	100.5 (94.0–116.0)	73.8 (81.5–87.6)	100.5 (90.3–110.5)	<0.0001	0.93
Start of TSST-C (HR0)	77.0 (73.5–80.5)	92.5 (87.8–107.0)	82.0 (70.1–89.9)	100.3 (94.5–104.4)	<0.0001	0.5
10 min after start of TSST-C (HR+10)	75.0 (65.8–81.3)	86.5 (81.3–101.3)	80.8 (72.9–89.1)	93.8 (85.1–98.63)	<0.0001	0.29
15 min after start of TSST-C (HR+15)	79.5 (76.8–85.3)	92.0 (82.8–103.0)	82.5 (72.6–87.8)	95.3 (85.1–101.0)	<0.0001	0.81
20 min after start of TSST-C (HR+20)	79.5 (74.3)	92.0 (84.0–98.5)	85.0 (76.0–92.3)	95.8 (83.3–99.9)	0.0001	0.31

Randomization group = physical activity versus sedentary activity; Weight status = overweight/obese versus normal weight; Data are shown as median (interquartile range); Differences between weight status and randomization groups are calculated by unpaired t test using the respective log values.

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OB compared to the NW group (p = 0.054, 95% *CI:* –0.001 to 11%) and its mean values over the 20 min after the TSST-C remained 5% higher (p = 0.03, 95% *CI:* 0.4–9%).

BP and HR Reactivity before the TSST-C

Adjusted S and D as well as HR at study begin (S/D/HR-70) did not differ between randomization arms or weight status (all $p \geq 0.9$) except for a higher S in the OW/OB compared to NW group (p = 0.02; Table 2 and Fig. 2b–d show unadjusted values).

Within the time course before the TSST-C, D and HR increased in the PA arm ($p \leq 0.02$) and S did not change (p = 0.8). In the sedentary arm, S and HR decreased ($p \leq 0.008$) and D did not change (p = 0.9)

Discussion

The novelty of this randomized study is the investigation of the role of acute PA in modulating both HPA and ANS reactivity to acute social stress in NW and also in OW/OB children. We found a significant interaction effect between the randomization arms and weight status for the HPA activation to stress exposure. These results are novel. In the NW children, the expected increase in the time course of cortisol in the sedentary arm was substantially reduced after PA with even a decrease in the cortisol concentration in this arm. In the OW/OB group, PA did not change the flat cortisol time course to the stress task. However, the mean cortisol concentrations after the stress task were different: In the OW/OB PA arm they were 80% and in the NW sedentary arm 49% higher than in the OW/OB sedentary arm. In general, for the total sample, cortisol levels increased significantly following the experimentally induced stress exposure, which shows that the stress manipulation was effective [46].

Further, the time course of the HR differed between the PA and sedentary arms with an increase in the sedentary and a decrease in the physically active children. The same tendency was observed for S, but not for D. Surprisingly the mean HR values after the stress task were higher in the PA arm. S was higher in the OW/OB compared to the NW children, both at study begin and also throughout the post-stress period.

The Impact of PA

Preceding acute PA increased cortisol reactivity to a stress test in the OW/OB, while it decreased it in the NW children. These results are novel in children and in the context of obesity. The results are in line with the study of Martikainen et al. [37] who investigated NW children with different levels of daytime PA representing chronic PA. They found decreased cortisol reactivity in NW children who were generally physically active. Both findings suggest that acute and chronic PA levels render the HPA axis more adaptive to acute stress in healthy children. Similarly, healthy adults engaging in high levels of chronic exercise show lower cortisol reactivity after a psychosocial stress task than those with low PA [29, 32, 33].

There was a tendency for changes in S values according to randomization arm, with increases in the sedentary and decreases in the PA arm. This result is in line with a lower BP during a post-stress condition after a previous acute exercise bout in healthy adults [52] and in children [38] and could be explained by a dampening of BP reactivity through the reduction of vascular resistance via b2-mediated vasodilation [52].

The time course of HR differed between the two activities. Despite the decrease in the HR after the stress task in the PA arm, the mean HR values remained higher in this arm. Our result might be partly explained by a deconditioning of the total sample, as greater physical fitness might decrease the PA and the stress-induced HR reactivity. Furthermore, the limited recovery period of 15 min between the exercise period and the beginning of the stress task

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in this study may have impeded on a full HR recovery. However, our results are in line with a previous study which also found differences in mean HR values after stress according to preceding PA in children [38]. A potential effect of higher body temperature after exercise cannot be excluded. In contrast, Bartholomew [31] found only differences regarding arterial BP, but not HR. We hypothesize therefore that acute exercise could possibly even be beneficial in this situation, as increased HR values after acute stress decreases the risk of developing obesity in healthy adults [53, 54]. There is evidence that heightened stress reactivity increases the risk for cardiovascular diseases [53]. However, blunted responses to stress are also associated with adverse health outcomes such as depression or obesity [27], which corresponds with the model of an inverted U-shape relationship between stress reactivity and health outcomes. This model suggests that low stress reactivity is linked to obesity or addiction and that high reactivity has a negative impact on cardiovascular health outcomes [26].

The Impact of Weight Status

The blunted cortisol reactivity to acute stress in the sedentary OW/OB children and the higher cortisol reactivity of sedentary NW children are in accordance with studies in obese adults. Obese adults show lower stress reactivity than lean adults [55] which confirms the inverse U-shape model (described above). Furthermore, levels of cortisol were higher in the OW/OB PA group, though the reactivity was not changed. Our findings might be explained by mitochrondrial dysfunction that has been linked to obesity [56] and has recently been related to psychological stress [57]. Picard et al. [57] showed that mitochondria can modulate stress responses via HPA axis and ANS reactivity. We conclude that cortisol hypo-responsiveness observed in obesity might also be linked to mitochondrial dysfunctions, and it might furthermore be linked to the activation of the reward system in the brain that is related to changes of food consumption and addiction [12] and may result in stimulated eating behavior and excessive weight gain [15].

A study on a selected low-income group of preschool children found similar associations of increased BMI (BMI z-scores) and blunted HPA stress reactivity [17]. This limited capacity to respond to challenging conditions might be the result of limited coping strategies, i.e. maladaptive or unhealthy strategies such as potential changes in food consumption to manage stress conditions [17, 58].

We also found that S and D at baseline and S after stress were higher in OW/OB children compared to NW children. This fits with previous data regarding higher BP levels in obese children, even at the prepubertal stage of development [55]. However, previous findings on the positive relationship of HR reactivity with body fat, BMI percentile and abdominal girth in children [23] were not confirmed in our sample. Potential methodological differences might be responsible for these discrepancies.

Impact of Previous Stress Exposure

Contrary to our predictions, OW/OB children had not been exposed to more stressful life events or early parental separation than NW children in this study. Several epidemiological studies had provided evidence that exposure to stressful life events is linked to obesity [9, 39]. However, our sample differs from those previous epidemiological studies, as it included a clinically obese population. Previous work of the authors [41] has shown that, within this pathological weight group, other stress conditions such as negative parenting style and pathological eating behavior are more prevalent in the OW/OB group and might therefore create chronic stress conditions that show more impact than major life events in these children.

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Study Strengths and Limitations

The strengths of the study are the stringent methodological design, including a clearly defined PA intervention, a standardized stress task specifically adapted to children, and a broad assessment of psychophysiological stress responses, including both branches of the stress system (the ANS and HPA axis) in NW and in OW/OB children. However, interpretation requires consideration of the methodological limitations of this study procedure. One limitation is related to the measurement of BP and HR during the laboratory session. The staff which measured BP and HR reactivities were not blind to the participants' allocation. However, to reduce the risk of a bias, all measurements were done using automatic machines and were always performed in the presence of another member of staff. Cortisol analyses were carried out in an independent laboratory by staff who were blind to the allocation. Secondly, the sample size was relatively small, and most OW/OB children were recruited from the clinics of the hospital, although sample characteristics were comparable to other studies [38, 46]. This might have had an impact on the power of the study. Further, it could be assumed that the NW sample was highly selective, but all participants of the NW sample were recruited through flyer advertisement or website advertisement of the same University Hospital of Lausanne where the OW/OB children were followed to capture participants with similar living conditions (urban, Swiss, similar exposure to more stressful life events or early parental separation). Descriptive values confirmed that 46% of the total sample showed low to moderate parental SES levels, and similar levels of 50% were found in the NW group. Results thus need further confirmation in a larger sample. Third, the small sample size and repeated testing might result in significant results that do not represent important effects. However, the main significant effects on the time course of cortisol ($p = 0.004$) and HR reactivity ($p = 0.006$) resist any correction for multiple testing.

Conclusion

This randomized study demonstrated for the first time that acute moderate PA can mitigate the reactivity of the HPA axis in response to stress. PA influenced stress reactivity differently in NW and OW/OB children. The cortisol increase after stress was reduced by PA in the NW children. However, cortisol levels were flat in the OW/OB children independently of their activity. On the other hand, mean cortisol levels after stress were higher in the OW/OB PA children compared to the sedentary OW/OB children. Mean cortisol levels were also higher in the sedentary NW children than in their sedentary OW/OB counterparts. PA also influenced ANS reactivity after the stress task. Children in the PA arm had higher mean HR values, but, in contrast to the children in the sedentary arm, their BP and HR decreased after stress.

PA may be seen as a protective factor, rendering healthy children less susceptible to the negative effects of and more adaptive to stress and the development of obesity.

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Disclosure Statement

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References

1 Ahrens W, Pigeot I, Pohlabeln H, De Henauw S, Lissner L, Molnár D, Moreno LA, Tornaritis M, Veidebaum T, Siani A; IDEFICS consortium. Prevalence of overweight and obesity in European children below the age of 10. Int J Obes. (Lond) 2014 Sep;38 Suppl 2:S99–107.
2 Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. JAMA. 2014 Feb;311(8):806–14.
3 Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. N Engl J Med. 2003 Apr;348(17):1625–38.
4 Michels N, Sioen I, Braet C, Eiben G, Hebestreit A, Huybrechts I, et al. Stress, emotional eating behaviour and dietary patterns in children. Appetite. 2012 Dec;59(3):762–9.
5 Vanaelst B, Michels N, Clays E, Herrmann D, Huybrechts I, Sioen I, et al. The association between childhood stress and body composition, and the role of stress-related lifestyle factors—cross-sectional findings from the baseline ChiBSD survey. Int J Behav Med. 2014 Apr;21(2):292–301.
6 Charmandari E, Tsigos C, Chrousos G. Endocrinology of the stress response. Annu Rev Physiol. 2005;67(1):259–84.
7 Gunnar M, Quevedo K. The neurobiology of stress and development. Annu Rev Psychol. 2007;58(1):145–73.
8 Heim C, Ehlert U, Hellhammer DH. The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. Psychoneuroendocrinology. 2000 Jan;25(1):1–35.
9 Koch FS, Sepa A, Ludvigsson J. Psychological stress and obesity. J Pediatr. 2008 Dec;153(6):839–44.
10 Michels N, Sioen I, Huybrechts I, Bammann K, Vanaelst B, De Vriendt T, et al. Negative life events, emotions and psychological difficulties as determinants of salivary cortisol in Belgian primary school children. Psychoneuroendocrinology. 2012 Sep;37(9):1506–15.
11 Gerke CK, Mazzeo SE, Stern M, Palmberg AA, Evans RK, Wickham EP 3rd. The stress process and eating pathology among racially diverse adolescents seeking treatment for obesity. J Pediatr Psychol. 2013 Aug;38(7):785–93.
12 Adam TC, Epel ES. Stress, eating and the reward system. Physiol Behav. 2007 Jul;91(4):449–58.
13 Kalon E, Hong JY, Tobin C, Schulte T. Psychological and Neurobiological Correlates of Food Addiction. Int Rev Neurobiol. 2016;129:85–110.
14 Smith DG, Robbins TW. The neurobiological underpinnings of obesity and binge eating: a rationale for adopting the food addiction model. Biol Psychiatry. 2013 May;73(9):804–10.
15 Sominsky L, Spencer SJ. Eating behavior and stress: a pathway to obesity. Front Psychol. 2014 May;5:434.
16 Yau YH, Potenza MN. Stress and eating behaviors. Minerva Endocrinol. 2013 Sep;38(3):255–67.
17 Miller AL, Clifford C, Sturza J, Rosenblum K, Vazquez DM, Kaciroti N, et al. Blunted cortisol response to stress is associated with higher body mass index in low-income preschool-aged children. Psychoneuroendocrinology. 2013 Nov;38(11):2611–7.
18 Hillman JB, Dorn LD, Loucks TL, Berga SL. Obesity and the hypothalamic-pituitary-adrenal axis in adolescent girls. Metabolism. 2012 Mar;61(3):341–8.
19 Lu Q, Tao F, Hou F, Zhang Z, Sun Y, Xu Y, et al. Cortisol reactivity, delay discounting and percent body fat in Chinese urban young adolescents. Appetite. 2014 Jan;72:13–20.
20 Ruttle PL, Javaras KN, Klein MH, Armstrong JM, Burk LR, Essex MJ. Concurrent and longitudinal associations between diurnal cortisol and body mass index across adolescence. J Adolesc Health. 2013 Jun;52(6):731–7.
21 Barnes VA, Treiber FA, Davis H, Kelley TR, Strong WB. Central adiposity and hemodynamic functioning at rest and during stress in adolescents. Int J Obes Relat Metab Disord. 1998 Nov;22(11):1079–83.
22 Goldbacher EM, Matthews KA, Salomon K. Central adiposity is associated with cardiovascular reactivity to stress in adolescents. Health Psychol. 2005 Jul;24(4):375–84.
23 Roemmich JN, Smith JR, Epstein LH, Lambiase M. Stress reactivity and adiposity of youth. Obesity (Silver Spring). 2007 Sep;15(9):2303–10.
24 Hursh BE, Fazeli MS, Wang S, Marchant EA, Woo P, Elango R, et al. Cardiac Autonomic Function at Baseline and under Stress and Its Relationship to Circulatory Markers of Inflammation in Obese Compared to Nonobese Children: A Pilot Study. Horm Res Paediatr. 2016;85(5):339–46.
25 Mazurak N, Sauer H, Weimer K, Dammann D, Zipfel S, Horing B, et al. Effect of a weight reduction program on baseline and stress-induced heart rate variability in children with obesity. Obesity (Silver Spring). 2016 Feb;24(2):439–45.
26 Carroll D, Ginty AT, Whittaker AC, Lovallo WR, de Rooij SR. The behavioural, cognitive, and neural corollaries of blunted cardiovascular and cortisol reactions to acute psychological stress. Neurosci Biobehav Rev. 2017 Jun;77:74–86.
27 Phillips AC, Ginty AT, Hughes BM. The other side of the coin: blunted cardiovascular and cortisol reactivity are associated with negative health outcomes. Int J Psychophysiol. 2013 Oct;90(1):1–7.
28 Glenney SS, Brockemer DP, Ng AC, Smolewski MA, Smolgovskiy VM, Lepley AS. Effect of Exercise Training on Cardiac Biomarkers in At-Risk Populations: A Systematic Review. J Phys Act Health. 2017 Dec;14(12):968–89.
29 Rimmele U, Zellweger BC, Marti B, Seiler R, Mohiyeddini C, Ehlert U, et al. Trained men show lower cortisol, heart rate and psychological responses to psychosocial stress compared with untrained men. Psychoneuroendocrinology. 2007 Jul;32(6):627–35.

30 Rimmele U, Seiler R, Marti B, Wirtz PH, Ehlert U, Heinrichs M. The level of physical activity affects adrenal and cardiovascular reactivity to psychosocial stress. Psychoneuroendocrinology. 2009 Feb;34(2):190–8.
31 Bartholomew JB. Stress reactivity after maximal exercise: the effect of manipulated performance feedback in endurance athletes. J Sports Sci. 2000 Nov;18(11):893–9.
32 Jackson EM, Dishman RK. Cardiorespiratory fitness and laboratory stress: a meta-regression analysis. Psychophysiology. 2006 Jan;43(1):57–72.
33 Spalding TW, Lyon LA, Steel DH, Hatfield BD. Aerobic exercise training and cardiovascular reactivity to psychological stress in sedentary young normotensive men and women. Psychophysiology. 2004 Jul;41(4):552–62.
34 Hamer M, Stamatakis E, Mishra G. Psychological distress, television viewing, and physical activity in children aged 4 to 12 years. Pediatrics. 2009 May;123(5):1263–8.
35 Parfitt G, Pavey T, Rowlands AV. Children's physical activity and psychological health: the relevance of intensity. Acta Paediatr. 2009 Jun;98(6):1037–43.
36 Griffiths LJ, Dowda M, Dezateux C, Pate R. Associations between sport and screen-entertainment with mental health problems in 5-year-old children. Int J Behav Nutr Phys Act. 2010 Apr;7(1):30.
37 Martikainen S, Pesonen AK, Lahti J, Heinonen K, Feldt K, Pyhälä R, et al. Higher levels of physical activity are associated with lower hypothalamic-pituitary-adrenocortical axis reactivity to psychosocial stress in children. J Clin Endocrinol Metab. 2013 Apr;98(4):E619–27.
38 Roemmich JN, Lambiase M, Salvy SJ, Horvath PJ. Protective effect of interval exercise on psychophysiological stress reactivity in children. Psychophysiology. 2009 Jul;46(4):852–61.
39 Gunstad J, Paul RH, Spitznagel MB, Cohen RA, Williams LM, Kohn M, et al. Exposure to early life trauma is associated with adult obesity. Psychiatry Res. 2006 May;142(1):31–7.
40 WHO | The WHO Child Growth Standards. https://www.who.int/childgrowth/en.
41 Horsch A, Wobmann M, Kriemler S, Munsch S, Borloz S, Balz A, et al. Impact of physical activity on energy balance, food intake and choice in normal weight and obese children in the setting of acute social stress: a randomized controlled trial. BMC Pediatr. 2015 Feb 19;15(1):12.
42 Ward DS, Bar-Or O. Use of the Borg scale in exercise prescription for overweight youth. Can J Sport Sci. 1990 Jun;15(2):120–5.
43 Rowland TW. Effects of obesity on aerobic fitness in adolescent females. Am J Dis Child. 1991 Jul;145(7):764–8.
44 Davies C, Godfrey S, Light M, Sakgeant A, Zeidifard E: Cardiopulmonary responses to exercise in obese girls and young wornen. J Appl Physiol. 1975 Mar;38(3):373–6.
45 Borg GA. Psychophysical bases of perceived exertion. Med Sci Sports Exerc. 1982;14(5):377–81.
46 Buske-Kirschbaum A, Jobst S, Wustmans A, Kirschbaum C, Rauh W, Hellhammer D. Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. Psychosom Med. 1997 Jul-Aug;59(4):419–26.
47 Bürgi F, Niederer I, Schindler C, Bodenmann P, Marques-Vidal P, Kriemler S, et al. Effect of a lifestyle intervention on adiposity and fitness in socially disadvantaged subgroups of preschoolers: a cluster-randomized trial (Ballabeina). Prev Med. 2012 May;54(5):335–40.
48 Pierrehumbert B, Ramstein T, Karmaniola A, Halfon O. Child care in the preschool years: Attachment, behaviour problems and cognitive development. Eur J Psychol Educ. 1996;11(2):201–14.
49 Obel C, Hedegaard M, Henriksen TB, Secher NJ, Olsen J, Levine S. Stress and salivary cortisol during pregnancy. Psychoneuroendocrinology. 2005 Aug;30(7):647–56.
50 Pluess M, Wurmser H, Buske-Kirschbaum A, Papousek M, Pirke KM, Hellhammer D, et al. Positive life events predict salivary cortisol in pregnant women. Psychoneuroendocrinology. 2012 Aug;37(8):1336–40.
51 Pruessner JC, Kirschbaum C, Meinlschmid G, Hellhammer DH. Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. Psychoneuroendocrinology. 2003 Oct;28(7):916–31.
52 Hamer M, Taylor A, Steptoe A. The effect of acute aerobic exercise on stress related blood pressure responses: a systematic review and meta-analysis. Biol Psychol. 2006 Feb;71(2):183–90.
53 Carroll D, Phillips AC, Der G. Body mass index, abdominal adiposity, obesity, and cardiovascular reactions to psychological stress in a large community sample. Psychosom Med. 2008 Jul;70(6):653–60.
54 Flaa A, Sandvik L, Kjeldsen SE, Eide IK, Rostrup M. Does sympathoadrenal activity predict changes in body fat? An 18-y follow-up study. Am J Clin Nutr. 2008 Jun;87(6):1596–601.
55 Farpour-Lambert NJ, Aggoun Y, Marchand LM, Martin XE, Herrmann FR, Beghetti M. Physical activity reduces systemic blood pressure and improves early markers of atherosclerosis in pre-pubertal obese children. J Am Coll Cardiol. 2009 Dec;54(25):2396–406.
56 Bournat JC, Brown CW. Mitochondrial dysfunction in obesity. Curr Opin Endocrinol Diabetes Obes. 2010 Oct;17(5):446–52.
57 Picard M, McManus MJ, Gray JD, Nasca C, Moffat C, Kopinski PK, et al. Mitochondrial functions modulate neuroendocrine, metabolic, inflammatory, and transcriptional responses to acute psychological stress. Proc Natl Acad Sci USA. 2015 Dec;112(48):E6614–23.
58 Lovallo WR. Do low levels of stress reactivity signal poor states of health? Biol Psychol. 2011 Feb;86(2):121–8.